Amendments to the Specification:

Please replace the paragraph beginning at page 3, line 25, with the following rewritten paragraph:

--FIG. 2 is an autoradiograph depicting the effect of anti-IFNAR2 mAbs on the ~~IGSF3~~ ISGF3 formation induced by type I IFN. ~~Hela~~ HeLa cells were first incubated with mAbs followed by the addition of IFN-α8 at a concentration of 20 ng/ml. Twenty minutes later, cell lysates were prepared and ~~IGSF~~ ISGF3 complex was detected by electrophoretic mobility shift assay. Anti-IFN-α mAb 9E1 was included as a positive control.--

Please replace the paragraph beginning at page 13, line 20, with the following rewritten paragraph:

--The anti-IFNAR2 antibodies of the invention can be made by using combinatorial libraries to screen for synthetic antibody clones with the desired activity or activities. In principle, synthetic antibody clones are selected by screening phage libraries containing phage that display various fragments of antibody variable region (Fv) fused to phage coat protein. Such phage libraries are panned by affinity chromatography against the desired antigen. Clones expressing Fv fragments capable of binding to the desired antigen are adsorbed to the antigen and thus separated from the non-binding clones in the library. The binding clones are then eluted from the antigen, and can be further enriched by additional cycles of antigen adsorption/elution. Any of the anti-IFNAR2 antibodies of the invention can be obtained by designing a suitable antigen screening procedure to select for the phage clone of interest followed by construction of a full length ~~anti4FNAR2~~ anti-IFNAR2 antibody clone using the Fv sequences from the phage clone of interest and suitable constant region (Fc) sequences described in Kabat et al., Sequences of proteins of Immunological Interest, Fifth Edition, NIH Publication 91-3242, Bethesda Md. (1991), vols. 1-3.--

Please replace the paragraph beginning at page 43, line 17 with the following amended paragraph:

--In another embodiment, the invention provides the anti-IFNAR2 monoclonal antibody produced by hybridoma cell line 1F3 (ATCC Deposit No. HB 12426).--

Please replace the paragraph beginning at page 43, line 19 with the following amended paragraph:

--In yet another embodiment, the invention provides the anti-IFNAR2 monoclonal antibody produced by hybridoma cell line 3B7 (ATCC Deposit No. HB 12427).--

Please replace the paragraph beginning at page 43, line 21 with the following amended paragraph:

--In an additional embodiment, the invention provides the anti-IFNAR2 monoclonal antibody produced by hybridoma cell line 1D3 (ATCC Deposit No. HB 12428).--

Please replace the paragraph beginning at page 63, line 21, with the following rewritten paragraph:

--~~Balb/c~~ BALB/c mice were immunized into each hind foot pad 11 times at two week intervals, with 2.5 μg of hIFNAR2-IgG fusion protein resuspended in MPL-TDM (Ribi Immunochem. Research Inc., Hamilton, Mont.). Three days after the final boost, popliteal lymph node cells were fused with murine myeloma cells, P3X63AgU.1 (ATCC CRL1597), using 35% polyethylene glycol. Hybridomas were selected in HAT medium. Ten days after the fusion, hybridoma culture supernatants were first screened for mAbs binding to the hIFNAR2-IgG fusion protein but not to CD4-IgG in a capture ELISA. The selected culture supernatants further tested for their ability to block the ligand-receptor binding in a capture ELISA as described below and for their ability to recognize cell membrane receptors on U266 cells by flow cytometric analysis as described in Chuntharapai et al., J. Immunol., 152:1783-1789 (1994). After cloning the selected final hybridomas twice, their antigen specificity as well as blocking activities were confirmed in the ligand-receptor binding assay, ~~IGSF-3~~ ISGF3 complex assay and anti-viral assay as described below.--

Please replace the paragraph beginning at page 69, line 20, with the following rewritten paragraph:

--The neutralizing abilities of these mAbs were determined in the receptor-ligand binding ELISA (FIG. 1). At a concentration of 0.6 nM (0.1 μg/ml), mAbs 1F3 and 3B7 were able to block greater than 90% of the biotinylated IFNα-2/1 binding to IFNAR2-IgG while even at a concentration of 6 nM (1 μg/ml), mAb 1D3 showed no significant blocking activity. From these ELISA results, it was determined that mAbs 1F3 and 3B7 are blocking mAbs and that mAb 1D3 may not be a blocking mAb. To further determine blocking activities of these mAbs, mAbs were tested in the ~~IGSF~~ ISGF3 complex formation EMSA and in the anti-viral assay. FIG. 2 depicts the results obtained in ~~IGSF~~ ISGF3 complex formation induced by IFN-α2 (IFN-αA) using L929 cells expressing hIFNAR2. At a concentration of 1 μg/ml, mAbs 3B7 and 1F3, but not mAb 1D3, completely blocked the ~~IGSF~~ ISGF3 complex formation induced by human type I IFN-α2. At a concentration of 10 μg/ml, all three mAbs blocked the ~~IGSF~~ ISGF3 complex formation. These results demonstrated that all three mAbs are blocking mAbs but mAb 1D3 is a weak blocking mAb. Blocking activities of these mabs on the anti-viral activities of type 1 IFNs (IFN α-1(D), -2(A), -5(G), -8(B), -2/1 and IFN-β) are summarized in Table 2 below.--

Please replace the paragraph beginning at page 78, line 8, with the following rewritten paragraph:

--The epitopes recognized by mAb 3B7 and mAb 1 D3 were localized to domain 1 (residues 49-55 and 68-72 in particular) and domain 2 (residues 133-139 and 153-157 in particular) IFNAR2, respectively. Although, the epitope(s) recognized by mAb 3B7 do not closely overlap with the epitope recognized by IFN-α2/1, mAb 3B7 exhibited the most potent blocking activity. In contrast, mAb 1D3 recognized residues located in lower domain 2 of IFNAR2, which forms a part of the ligand binding areas and showed weak blocking activities in the ~~ISGF~~ ISGF3 complex formation and in anti-viral assay.--